Ergot: Biology and Control

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February, 2006

Introduction

When we look at the tens of thousands of known species of fungi, few have had a greater impact on society than ergot. Over a thousand compounds have been extracted or derived from ergot, and no other natural product has been of greater value to the pharmaceutical industry. Drugs in obstetrics, neurology, and psychology, for example, are derived from ergot. One of the best known of these derivatives is LSD. However, consumption of raw ergot can result in ergot poisoning, often referred to as ergotism. Some of the symptoms of ergotism include hallucinations, severe pain, convulsions, gangrenous limbs and death. In the middle ages, ergotism resulting from consumption of bread made from ergot contaminated flour was referred to as St. Anthony's fire. Ergot has a long and interesting history. And in recent years, ergotism has even been linked to the erratic behavior of some individuals in 1692, as described in the Salem witchcraft trials, although this association has been disputed.

The earliest mention of ergot is in a German herbal from 1582, where we find the first recorded medical use of ergot, as an aid in childbirth. The first illustration of ergot appears in 1658. Although the medicinal uses of ergot were known in the middle ages, the risk of ingestion of bread made from ergot contaminated rye flour was somehow not recognized. This was especially unfortunate for the impoverished peasants, who relied on the highly susceptible rye as their primary source for bread. Reports of ergot poisonings in humans extend from the Middle Ages into modern times, including a report from Manchester in 1929 and Ethiopia in 1979. Reports of ergotism in animals appear annually in the veterinary literature. Despite the long history of ergot, it was not until 1853 that ergot was recognized as a disease, caused by a fungus, and not simply a malformation of the plant. And despite over 150 years of research on ergot, the disease continues to plague growers of cereal grains and grasses. As recently as 2005, we find reports of widespread occurrence of ergot in barley in the Midwest. Ergot continues to be problematic in Kentucky bluegrass seed production in the Pacific Northwest.

General overview of ergot

The term ergot is the common disease name for a group of fungi in the genus *Claviceps*. Ergot also refers to the typically elongated fungal structure, technically known as a sclerotium (Figure 1). *Claviceps* is a unique group of fungi, which infect only the ovaries of grasses, replacing what would normally be a seed with a sclerotium. There are some 40 known species of *Claviceps*. Most are tropical or subtropical and nearly all are restricted to a single genus or closely related genera. Ten species occur in the U.S., but only three are of economic importance. The recently introduced *Claviceps africana* (Figure 2) is a serious pathogen of sorghum in the Midwest and south central states. *Claviceps paspali* (Figure 3) is an important pathogen in seed production of *Paspalum* species in the southern states. But the species of greatest concern is the widespread and common *Claviceps purpurea* (Figure 4). This species is unique in having an exceptionally broad host range, including as many as 400 species of grasses, including wheat, rye, barley, and all of the cool season forage grasses.

Figure 1. Sclerotia of Claviceps purpurea.

Figure 2. *Claviceps africana* on sorghum

Figure 3. *Claviceps paspali* on dallisgrass

Figure 4. *Claviceps purpurea* on fine fescue

Biology of Ergot

The sclerotium of *Claviceps pupurea* is a specialized structure (Figure 5). The outer layer, or rind, provides protection and resists attack from insects and other microbes. The internal tissue is high in fats and nutrients needed to support germination and production of fruiting bodies. The sclerotium overwinters on or in the soil and will not germinate until dormancy within the sclerotium is broken by a period of several weeks of cold temperatures.

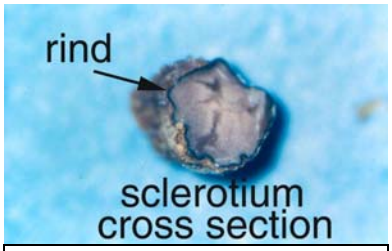

Figure 5. Cross section of a sclerotium.

Germination begins in the spring. Moist soil is required for germination of sclerotia. Dry conditions will interrupt germination, but it will proceed when wet conditions resume.

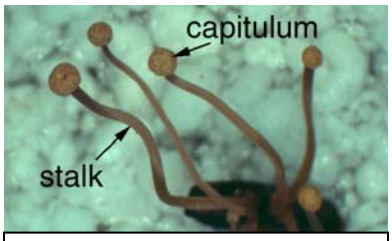

Figure 6. Fruiting bodies of *Claviceps purpurea*.

The early phase of germination and fruiting of the sclerotium includes production of a stalk (Figure 6). The stalk will be short if the sclerotium is on the soil surface, but if below the surface or under leaf litter, the stalk will continue to elongate, up to 1 to 2 inches, until it reaches light. A specialized spherical structure called a capitulum, no bigger than the head of a pin, develops at the end of the stalk. Embedded within the outer surface of the capitulum are spherical or flask shaped structures called perithecia (Figure 7). Within each perithecium are numerous slender bodies called asci. Each ascus contains 8 ascospores (Figure 8).

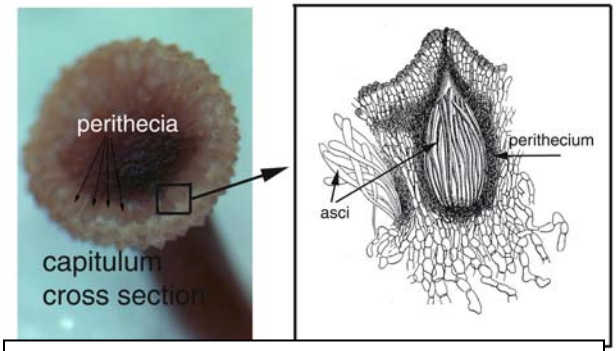

Figure 7. Perithecia in capitulum and cross section of perithecium showing asci.

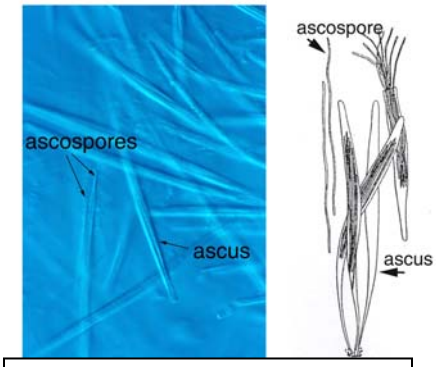

Figure 8. Asci and ascospores.

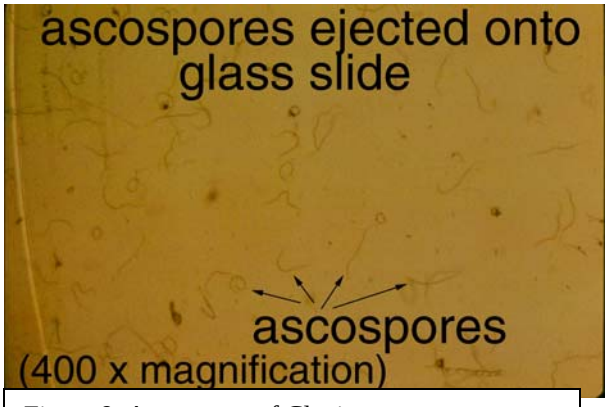

Figure 9. Ascospores of Claviceps purpurea ejected onto glass slide.

Considering the number of asci within each perithecium and the number of perithecia within each capitulum, each capitulum is capable of producing thousands of ascospores. The slender thread-like ascospores are ejected from the perithecia, typically early in the morning, and the microscopic spores, finer than dust particles, are transported with only the slightest movement of air. The production and release of ascospores occurs when there is a high moisture content at the soil surface, typically as the result of rain or irrigation. Given a rain event, ascospore production will often continue for several days, until the soil surface dries. Ascospore production continues when the soil is once again moist.

Figure 10. Seed and sclerotia from annual ryegrass and Kentucky bluegrass.

The size of the sclerotium that develops is determined by the size of the seed of the host plant. Small seeded grasses such as bentgrass or bluegrass produce much smaller sclerotia than large seeded grasses such as wheat and barley. The larger the sclerotium, the more stalked capitula, or fruiting bodies, that will be produced. Kentucky bluegrass sclerotia typically produce 3 to 6 fruiting bodies. Six to twelve fruiting bodies would be expected from a large sclerotium. Sclerotia do not all germinate at the same time. Some will germinate early, some late. This ensures ascospore production over a several month period in the spring, beginning with early flowering grasses.

Figure 11. Stigmas of annual ryegrass.

The timing of ascospore production coincides with flowering in grasses. The feathery stigmas, so effective in trapping airborne pollen, are equally effective in collecting ascospores. The ovary is the only organ of the grass plant susceptible to infection and the fungus will not infect any other part of the plant. In most cases, the fungus enters at the base of the ovary and begins to colonize the tissue. Within a few days of infection a sphacelium, or surface producing spores, develops. As the spores, called conidia, are produced they mix with plant sap leaking from the infection site in what is commonly referred to as the honeydew stage.

Figure 12. Honeydew on Kentucky bluegrass.

As the sap evaporates, sugars and conidia are concentrated into a sticky syrup. The high sugar content prevents conidia from germinating. However, conidia that are transferred to other flowers, especially unfertilized flowers, can infect those flowers. Rainy and windy conditions can be effective in spreading the conidia to other flowers, contributing to additional infections. About 7 to 10 days after infection, the outer sphacelium is converted into the rind of the sclerotium and the structure begins to take on the characteristics of the sclerotium.

It is important to keep in mind that the longer the duration of flowering the longer the duration that the grass will be susceptible to infection, and the greater the probability of infection. Cool conditions, and especially cool wet conditions that prolong the flowering period, will favor ergot infection and greater disease severity. It is also important to keep in mind that although moisture is required for ascospore production, such conditions are not required for infection from honeydew. In fact, ergot can continue to develop under warm, dry conditions if honeydew is transferred to uninfected flowers. This can occur through head to head contact, movement by insects, or any other means, animal, human, or mechanical that transfers the sticky honeydew from one flower to

another. The honeydew is very dense with conidia and even the tiniest amount is enough to infect a flower.

During the past 100 years we have made some progress in our understanding of ergot although disease management continues to be a challenge. There are several approaches to management of ergot, including disease resistance, fungicides, and minimizing primary and secondary sources of inoculum.

Ergot control

Disease Resistance

In the cereal grains, breeding for disease resistance has proven very effective in ergot management. In the cereal grains, resistance to infection of an ovary generally develops within several days after fertilization. In some cultivars of barley, resistance to ergot can develop as soon as several hours after fertilization. Since flowers are the only organ of the plant susceptible to infection, cultivars with a short flowering period are desirable. In Kentucky bluegrass it is not known whether resistance to ergot infection develops soon after fertilization. However, this question is being addressed at the USDA national forage seed production research center in Corvallis. We do know that some cultivars of Kentucky bluegrass consistently develop less disease than others. This differential susceptibility has also been verified under controlled greenhouse studies in research labs here and in Europe. However, the nature or mechanism of this resistance has not been elucidated.

Fungicides

Several studies have demonstrated that properly timed fungicide applications can reduce the severity of ergot in Kentucky bluegrass. For optimum effect, the fungicides must be applied at the beginning of flowering. Although fungicides reduce the severity of ergot, it is not clear whether there is an economic benefit from fungicide application.

Alternate sources of inoculum

In the cereal grains, weed grasses within or surrounding fields are known to be a source of ergot inoculum. Control of weeds through sprays, or by cutting grasses surrounding fields to prevent heading is recommended. In the Willamette Valley, ergot can develop very early in the spring on annual bluegrass, which is one of the first grasses to flower in the spring. Infected bluegrass at the honeydew stage can be a source of inoculum for later flowering grasses. In addition, sclerotia produced by annual bluegrass and other weed grasses will rest in the soil, dormant until the following spring, when ascospores from these sclerotia become an important source of inoculum.

Sclerotia at harvest

Since sclerotia are the primary source of inoculum, removal of as many sclerotia as possible during harvest reduces the potential number of ascospores the following spring. The conidia do not survive, and the fungus does not grow or survive on grass residues. It is only the scleotia that overwinter and produce ascospores in the spring. The greater the number of sclerotia removed from the field during harvest, the fewer remaining for ascospore production.

Burning

Some reduction in ergot will be realized through post harvest open field burning. Propane burning may also be of some benefit, providing that temperatures at the soil surface are high enough to incinerate the sclerotia.

Vacuum Sweeping

Vacuum sweeping was used in some fields in the Willamette Valley as a post harvest alternative to field burning. However, the efficiency of the vacuum sweep machine in removal of sclerotia from a field has not been established.

Composting

In trials in the Willamette valley, sclerotia did not survive composting of post-harvest grass straw. In these trials temperatures as high as 150 F were recorded.

Mulching.

The effect of mulches on ergot survival or fruiting bodies had not been investigated.

Insect control

Insects, especially flies and moths are attracted to ergot honeydew as a food source. As these insects crawl around a seed head they can pick up and transfer honeydew to other flowers, contributing to disease spread. However, quantitative information on the role of insects in ergot epidemiology is lacking. It is not clear how efficient insects are in secondary spread of ergot. It is not known if control of insects would reduce secondary spread of ergot. In a recent study of insects and ergot in Central Oregon there was no correlation between number of insects within fields and severity of ergot.

Irrigation.

Sprinkler irrigation at the beginning of flowering can support ascospore production for primary infections. However, it is not know whether irrigation would facilitate secondary spread of ergot.

Row spacing / orientation

It is not known whether a closed canopy would block ascospore movement from the soil to the seed heads or whether wider row spacing would provide more rapid drying at the soil surface to reduce ascospore production.

It is not know whether orientation of rows in line with prevailing winds would have any effect on ergot severity. For some fungal pathogens, orientating rows in line with prevailing winds shortens the duration of leaf wetness, resulting in less disease severity.

Future outlook

Long term control of ergot will be best achieved though programs to improve ergot resistance. There has been considerable progress in this direction in recent years. In addition, management practices to reduce the population of ergot sclerotia in the field

though control of weed grasses, removal of sclerotia during harvest, post harvest burning, or other means will help to reduce ergot severity. There are some unknowns, such as the effect of row spacing or orientation on ergot severity. Research in this area may be helpful. A more significant question, however, is whether the timing and duration of irrigation would affect ergot severity. Given the importance of soil surface moisture content at the beginning of flowering, timing of irrigation may a means to reduce the onset or severity of ergot.